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1: [Deng WG, Saunders MA, Gilroy DW, He XZ, Yeh H, Zhu Y, Shtivelband MI, Ruan KH, Wu KK.](#) Related Articles, Li

Purification and characterization of a cyclooxygenase-2 and angiogenesis suppressing factor produced by human fibroblasts. *FASEB J.* 2002 Aug;16(10):1286-8. *Epub 2002 Jun 07.* PMID: 12060668 [PubMed - indexed for MEDLINE]

2: [Danz H, Stoyanova S, Wippich P, Brattstrom A, Hamburger M.](#) Related Articles, Li

Identification and isolation of the cyclooxygenase-2 inhibitory principle in *Isatis tinctoria*. *Planta Med.* 2001 Jul;67(5):411-6. PMID: 11488453 [PubMed - indexed for MEDLINE]

3: [Abad T, McNaughton-Smith G, Fletcher WQ, Echeverri F, Diaz-Penate R, Tabraue C, Ruiz de Galarreta CM, Lopez-Blanco F, Luis JG.](#) Related Articles, Li

Isolation of (S)-(+)-naproxene from *Musa acuminata*. Inhibitory effect of naproxene and its 7-methoxy isomer on constitutive COX-1 and inducible COX-2. *Planta Med.* 2000 Jun;66(5):471-3. PMID: 10909271 [PubMed - indexed for MEDLINE]

4: [Percival MD, Ouellet M, Vincent CJ, Yergey JA, Kennedy BP, O'Neill GP.](#) Related Articles, Li

Purification and characterization of recombinant human cyclooxygenase-2. *Arch Biochem Biophys.* 1994 Nov 15;315(1):111-8. PMID: 7979387 [PubMed - indexed for MEDLINE]

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1: Proc Natl Acad Sci U S A. 1992 Aug 15;89(16):7384-8.

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Human cyclooxygenase-2 cDNA.

Hla T, Neilson K.

Department of Molecular Biology, Holland Laboratory, American Red Cross, Rockville MD 20855.

Cyclooxygenase (Cox), also known as prostaglandin (PG) H synthase (EC 1.14.99.1), catalyzes the rate-limiting step in the formation of inflammatory PGs. A major regulator step in PG biosynthesis is at the level of Cox: growth factors, cytokines, and tumor promoters induce Cox activity. We have cloned the second form of the Cox gene (Cox-2) from human umbilical vein endothelial cells (HUVEC). The cDNA encodes a polypeptid of 604 amino acids that is 61% identical to the previously isolated human Cox-1 polypeptide. In vitro translation of the human (h)Cox-2 transcript in rabbit reticulocyte lysates resulted in the synthesis of a 70-kDa protein that is immunoprecipitated by antiserum to ovine Cox. Expression of the hCox-2 open reading frame in Cos-7 monkey kidney cells results in the elaboration of cyclooxygenase activity. hCox-2 cDNA hybridizes to a 4.5-kilobase mRNA species in HUVEC, whereas the hCox-1 cDNA hybridizes to 3- and 5.3-kilobase species. Both Cox-1 and Cox-2 mRNAs are expressed in HUVEC, vascular smooth muscle cells, monocytes, and fibroblasts. Cox-2 mRNA was preferentia induced by phorbol 12-myristate 13-acetate and lipopolysaccharide in human endothelia cells and monocytes. Together, these data demonstrate that the Cox enzyme is encoded b at least two genes that are expressed and differentially regulated in a variety of cell types. High-level induction of the hCox-2 transcript in mesenchymal-derived inflammatory cel suggests a role in inflammatory conditions.

PMID: 1380156 [PubMed - indexed for MEDLINE]

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ACCESSION NUMBER: 1994-46604 DRUGU B P E
TITLE: Characterization of the mechanism of inhibition of
human cyclooxygenase-2 by
anti-inflammatory drugs.
AUTHOR: Ouellet M; Percival M D
CORPORATE SOURCE: Merck-Frosst
LOCATION: Kirkland, Quebec, Canada
SOURCE: Can.J.Physiol.Pharmacol. (72, Suppl. 1, 453, 19 1 Ref.
CODEN: CJPPA3 ISSN: 0008-4212
AVAIL. OF DOC.: Merck Frosst Centre for Therapeutic Research, Kirkland, QC
H9R 4P8, Canada.
LANGUAGE: English
DOCUMENT TYPE: Journal
FIELD AVAIL.: AB; LA; CT
FILE SEGMENT: Literature
AB The kinetic mechanism of inhibition of the **purified** form of
inducible cyclooxygenase (hCox-2) by several classical NSAIDs and a
selective Cox-2 inhibitor was determined. Flurbiprofen, meclofenamate
and indometacin were all time-dependent inhibitors of hCox-2. None of
the 3 inhibitors had a high degree of selectivity, when compared with
hCox-1. NS-398 also had time-dependent inhibition of hCox-2, but was a
time-independent inhibitor of hCox-1. The difference in the mechanism of
inhibition was reflected in the high degree of selectivity observed for
hCox-2 over hCox-1. Results demonstrate that the mechanism of inhibition
of hCox-2 by classical NSAIDs is similar to that identified for ovine
Cox-1. In addition, it shows the nature of time-dependency of inhibition
of hCox-1 and hCox-2 greatly determines the degree of selectivity for 1
isozyme over the other. (conference abstract).